

Fluid De-escalation Strategy in Managing Critically Ill Patients: A Narrative Review

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Abstract:

Fluid therapy is a mainstay in the management of critically ill patients with sepsis, shock, trauma, and perioperative states. While early aggressive resuscitation is often life-saving, excessive fluid administration can lead to fluid overload, associated with worse clinical outcomes. To address this, the concept of fluid stewardship has emerged—viewing fluid as a drug with indications, dosing, and toxicities. Within this, the ROSE model (Resuscitation, Optimization, Stabilization, Evacuation) conceptualizes the dynamic phases of fluid therapy, with de-escalation (deresuscitation) being crucial but often under-recognized. This narrative review (~4000 words) explores the physiological basis, clinical strategies, monitoring, evidence, and challenges of fluid de-escalation across various patient populations. It concludes with future directions to better integrate the de-escalation phase into critical care practice.

Key words: De-escalation, Fluid management, intensive care unit, sepsis

Introduction:

Intravenous fluids are administered to nearly all critically ill patients, particularly those in shock, to restore intravascular volume, improve tissue perfusion, and reduce organ failure risk. Historically, fluid management was aggressive, a “fill-the-tank” approach rooted in early resuscitation needs.^[1] However, over the past two decades, data have revealed that persistent positive fluid balance contributes to pulmonary edema, delayed mechanical ventilation weaning, intra-abdominal hypertension, impaired wound healing, and increased mortality.^[2–4] This led to the concept of fluid stewardship, treating intravenous fluids like pharmacotherapy with clear indications, dosing limits, and tapering protocols.^[5]

Derived from these insights is the ROSE model, which divides fluid therapy into four phases: Resuscitation, Optimization, Stabilization, and Evacuation (de-escalation). This model reframes fluid therapy as dynamic, where initial support transitions to cautious restriction and active removal as stability returns.^[6–8] Despite this, de-escalation remains inconsistently applied due to fears of hypoperfusion, hemodynamic instability, or renal compromise. This

comprehensive review aims to synthesize the pathophysiological rationale, clinical strategies, evidence base, patient-specific considerations, and implementation barriers surrounding fluid de-escalation.

Evolution of Fluid Therapy and the ROSE Model

The dominant paradigm for decades endorsed liberal fluid administration to reverse shock and restore perfusion. However, pivotal trials and observational data challenged this view. The ARDSNet FACTT trial compared liberal versus conservative fluid strategies in ARDS and found that a conservative approach improved lung function, increased ventilator-free days, without increasing shock or renal failure.^[9] Observational studies across sepsis, trauma, burns, and postoperative populations consistently linked positive fluid balance with worse outcomes.^[3,10]

To articulate this nuanced progression, Malbrain and colleagues formalized the ROSE model.^[6] This acknowledges that fluids are life-saving early (R), but as stability emerges (O, S), continued liberal use becomes detrimental, necessitating the Evacuation phase. Vincent and De Backer offered a similar SOSD (Salvage, Optimization, Stabilization, De-escalation) framework. These models underscore the need to pivot from resuscitation to deresuscitation as soon as feasible.^[7,8]

Pathophysiological Basis of Fluid Overload and De-escalation

Critical illness often prompts systemic inflammation and endothelial dysfunction, resulting in capillary leakage and extravascular fluid accumulation. The resulting interstitial edema impairs organ function through diverse mechanisms. Increased extravascular lung water leads to alveolar flooding, reduced compliance, delayed ventilator weaning, and worsened oxygenation.^[10] Raised interstitial and venous pressure reduces glomerular perfusion, contributing to oliguria and worsening AKI.^[11] Fluid overload exacerbates myocardial stretch in patients with limited cardiac reserve, worsening function.^[12] Edema increases intra-abdominal pressure, compromising splanchnic perfusion, renal function, and increasing risk of abdominal compartment syndrome.^[13] Fluid de-escalation (deresuscitation) aims to remove this harmful excess via active strategies (diuretics, ultrafiltration) or through early negative fluid balance. This restores euvolemia, enhances organ recovery, and shortens ICU stays—but only when performed once patients are hemodynamically stable.^[14,15]

Strategies for Fluid De-escalation

Conservative fluid management: Once resuscitation targets are met, fluids should be minimized. Maintenance infusions should be halted or limited, and routine boluses avoided unless strictly indicated. In hypotensive patients, vasopressors are preferred over additional fluids.^[5] The focus shifts from “fluid responsiveness” to “fluid tolerance”—assessing whether further fluid could cause harm rather than help.^[16]

Active fluid removal: diuretics: Loop diuretics (e.g., furosemide) remain the most accessible tool for deresuscitation, facilitating natriuresis and diuresis. Continuous infusion may overcome diuretic resistance, while adjunctive thiazides or acetazolamide can potentiate effect.^[17,18] Risks include electrolyte imbalance, hypotension, and worsened renal function—necessitating close monitoring.

Renal replacement therapy (RRT): In cases of refractory overload or concurrent AKI, RRT—especially continuous modalities like CVVH or CVVHDF—can gently remove fluid while mitigating hypotension risk. Optimal ultrafiltration rates lie between 1.0–1.75 mL/kg/h; rates outside this risk under-treatment or organ hypoperfusion.^[19,20] Monitoring preload status is vital; loss of preload responsiveness during removal is a signal to pause.^[20]

Adjunctive maneuvers: Adjuncts such as albumin (to redistribute interstitial fluid into the vascular compartment), passive leg raising, and early mobilization can enhance deresuscitation. This is especially useful when diuretic response is inadequate and RRT is not yet indicated.^[5,21]

Monitoring during fluid de-escalation

Ensuring safe and effective deresuscitation requires vigilant monitoring like blood pressure, heart rate, urine output, mental status, lactate, capillary refill time. In certain cases advanced monitoring like echocardiography, dynamic metrics (pulse-pressure variation, stroke volume variation), extravascular lung water, and bioimpedance measurements are considered. This will be subjective to availability of facilities.^[22] Cumulative daily fluid balance, body weight changes—especially useful in resource-constrained settings are simple alternative methods to monitor.^[23] A cautious, individualized approach based on fluid tolerance rather than responsiveness improves safety.

Evidence from trials and observational studies

The body of evidence supporting fluid de-escalation has grown substantially over the past two decades, though high-quality randomized controlled trials remain relatively limited compared to studies on initial resuscitation. Among the landmark investigations, the Fluid and Catheter Treatment Trial (FACTT) conducted by the ARDS Network remains the most influential randomized controlled trial in this field. Involving over 1,000 patients with acute lung injury/ARDS, the trial compared a conservative fluid management strategy with a more liberal approach. Results showed that conservative management was associated with significantly improved pulmonary outcomes, including better oxygenation and reduced extravascular lung water, and importantly, a shorter duration of mechanical ventilation and ICU stay. While there was no statistically significant mortality difference, the improvement in respiratory outcomes without harm cemented the role of conservative strategies in patients with acute lung injury.^[9] These findings were reinforced by a large systematic review and meta-analysis that synthesized data from nearly 20,000 critically ill patients across multiple observational and interventional studies. The analysis revealed that non-survivors consistently demonstrated a greater positive cumulative fluid balance, averaging approximately 4.4 L more after one week compared to survivors. Furthermore, patients managed with restrictive or deresuscitative strategies exhibited a significantly reduced mortality risk, with a pooled odds ratio of 0.42, suggesting a nearly 60% relative risk reduction.^[3] This provided compelling population-level evidence that excess fluid accumulation is not merely an epiphenomenon of disease severity but a modifiable risk factor.

More recently, the RADAR-2 feasibility randomized controlled trial explored whether combining conservative fluid administration with active deresuscitation could be safely and effectively implemented in critically ill patients. Patients randomized to the intervention arm achieved a significantly lower net fluid balance by day 3 (–840 mL) compared to those

managed with usual care (+130 mL), with statistical significance ($P < 0.01$). Although not powered to detect mortality differences, the trial demonstrated proof of concept that a structured fluid stewardship approach incorporating deresuscitation is feasible, safe, and able to achieve negative balance within days of ICU admission.^[24]

Complementing trial data, large cohort studies have provided important real-world insights. In the RADAR observational cohort study, Silversides and colleagues evaluated fluid balance trajectories in critically ill patients. They found that achieving a negative balance by day 3—whether spontaneously or through active deresuscitation—was independently associated with markedly improved survival. Specifically, negative balance was linked to an approximately 80% reduction in 30-day mortality (odds ratio ~0.2), underscoring the prognostic significance of fluid removal once the acute resuscitative phase has passed.^[25,26]

Further synthesis comes from meta-analyses focused on sepsis and ARDS populations. These analyses consistently demonstrate that conservative or deresuscitative strategies confer important clinical benefits, particularly in improving ventilator-free days and reducing ICU length of stay. However, effects on overall mortality have been less consistent, with some analyses showing benefit and others failing to reach statistical significance, likely reflecting study heterogeneity, variable definitions of deresuscitation, and differences in timing of intervention.^[27]

The most up-to-date expert consensus is encapsulated in the 2025 ESICM Fluid Management Guidelines (Part 3). These guidelines, based on systematic review of 13 RCTs, issue a conditional recommendation in favor of fluid de-escalation strategies compared with no de-escalation, recognizing the overall balance of evidence and clinical experience. Furthermore, they specifically recommend diuretic-guided deresuscitation over usual care, given moderate-certainty evidence for efficacy and safety. At the same time, the guidelines caution against the use of ultrafiltration in the absence of other renal replacement therapy indications, citing insufficient benefit and potential harm in this context.^[2]

Taken together, this body of evidence suggests that fluid de-escalation is both feasible and beneficial across diverse critically ill populations. While ongoing large RCTs are needed to define optimal timing, patient selection, and modalities, current trial, observational, and guideline data strongly support integrating de-escalation into modern critical care fluid stewardship.

Clinical Applications in Specific Populations

While the principles of fluid de-escalation are broadly relevant to all critically ill patients, their application must be tailored to the underlying pathophysiology and clinical context. Different populations—such as patients with sepsis, acute respiratory distress syndrome (ARDS), trauma, burns, postoperative surgical states, and those with renal or cardiac comorbidities—have unique risks associated with fluid accumulation, and correspondingly, varying thresholds and strategies for deresuscitation.

Sepsis and Septic Shock

In patients with sepsis, particularly septic shock, the initial phase of fluid therapy is dominated by aggressive resuscitation to correct life-threatening hypoperfusion. However, once hemodynamic stability is achieved, persistence of positive fluid balance has been strongly associated with worse outcomes, including impaired oxygen delivery, tissue edema, and

increased mortality. Observational studies have shown that a negative fluid balance achieved within the first 72 hours is independently associated with improved survival. Conservative fluid management, combined with early vasopressor support to maintain perfusion, is now considered a cornerstone of sepsis care. De-escalation in this context often involves diuretic-assisted deresuscitation or renal replacement therapy (RRT) in cases of concomitant acute kidney injury (AKI). The challenge lies in distinguishing ongoing sepsis-induced vasoplegia from hypovolemia, underscoring the importance of advanced hemodynamic monitoring to guide decisions.^[10,27]

Acute Respiratory Distress Syndrome (ARDS)

ARDS is one of the populations where fluid de-escalation strategies have been most rigorously studied. Extravascular lung water contributes directly to hypoxemia, reduced compliance, and prolonged ventilator dependence. The FACTT trial provided high-level evidence that conservative fluid management improves pulmonary outcomes without increasing renal failure risk. In clinical practice, deresuscitation in ARDS patients often becomes a priority once oxygenation is stabilized with lung-protective ventilation and perfusion goals are met. Diuretics are typically the first-line approach, with continuous infusion favored over boluses in unstable patients. Importantly, clinicians must balance the benefits of improved pulmonary function with the risk of compromising right ventricular preload, particularly in patients with pulmonary hypertension or right ventricular dysfunction.^[9,27]

Trauma and Hemorrhagic Shock

Fluid management in trauma patients follows a different trajectory due to the initial emphasis on damage control resuscitation, where restrictive fluid strategies and permissive hypotension are often employed until hemostasis is achieved. However, after the acute bleeding phase is controlled, trauma patients frequently accumulate significant fluid loads due to massive transfusions, crystalloids, and capillary leak from systemic inflammation. This “second hit” phenomenon, compounded by resuscitation practices, makes trauma patients particularly susceptible to abdominal compartment syndrome, pulmonary edema, and multiorgan dysfunction. Fluid de-escalation in this population requires careful timing, often beginning after hemorrhage control, with aggressive monitoring of intra-abdominal pressure, lung function, and renal perfusion to avoid iatrogenic harm.^[28,29]

Major Surgery and Postoperative Patients

In the perioperative setting, particularly in patients undergoing major abdominal, thoracic, or vascular surgery, the risk of fluid overload is amplified by intraoperative administration of crystalloids, colloids, and blood products. Excessive fluid administration has been linked with anastomotic leaks, delayed wound healing, pulmonary complications, and prolonged hospital stay. Enhanced Recovery After Surgery (ERAS) protocols emphasize restrictive and individualized fluid therapy, including early deresuscitation strategies postoperatively. Conservative use of fluids, guided by dynamic measures of fluid responsiveness and postoperative monitoring, has been shown to reduce complications and accelerate recovery. Postoperative fluid de-escalation is particularly crucial in patients with cancer, as fluid overload has been associated with impaired immune function and increased risk of tumor recurrence.^[30]

Burn Injury

Patients with severe burns represent a unique challenge in fluid management due to the profound capillary leak and evaporative losses during the first 24–48 hours. The Parkland formula has traditionally guided resuscitation, but excessive crystalloid administration—termed “fluid creep”—can result in devastating complications, including compartment syndromes, pulmonary edema, and worsening tissue ischemia. Once the initial resuscitation phase has been completed and capillary integrity begins to restore, burn patients often require aggressive fluid de-escalation to mitigate the effects of iatrogenic overload. Deresuscitation in this group typically involves a combination of diuretics, albumin-assisted fluid mobilization, and close monitoring of renal and cardiovascular function. The timing is critical, as premature deresuscitation may compromise perfusion in patients still experiencing capillary leakage.

Cardiac Failure and Cardiorenal Syndrome

In critically ill patients with pre-existing heart failure or those developing acute decompensated heart failure during ICU stay, fluid overload has particularly detrimental effects. Venous congestion worsens pulmonary edema, impairs hepatic and renal function, and reduces tolerance to enteral feeding. Deresuscitation is especially crucial but also more complex in this group, as aggressive fluid removal can precipitate hypotension or worsen renal dysfunction. Strategies often involve cautious use of loop diuretics, sometimes in combination with thiazides or ultrafiltration in resistant cases. Monitoring of central venous pressure, echocardiography-derived filling pressures, and bioimpedance techniques can guide safe de-escalation.

Acute kidney injury and renal replacement therapy

In patients with AKI, fluid overload is not only a marker of disease severity but also an independent contributor to poor outcomes. Observational studies have consistently demonstrated that fluid overload at the initiation of RRT is associated with increased mortality. In these patients, RRT serves not only as a renal support modality but also as a powerful tool for deresuscitation, allowing controlled ultrafiltration to achieve negative balance. Continuous RRT modalities are generally favored in hemodynamically unstable patients, as they permit gradual removal of excess fluid. Careful coordination with the timing of nutrition, drug administration, and hemodynamic support is essential to maximize benefit while minimizing risks of hypoperfusion.

Practical challenges and controversies

Despite the growing body of evidence supporting fluid de-escalation, its implementation in clinical practice remains fraught with challenges and controversies. Clinicians frequently struggle with the balance between optimizing organ perfusion and avoiding the harms of fluid overload. Unlike the initial resuscitative phase, where the goals are well defined (i.e., restoration of perfusion in shock), the transition to de-escalation lacks clear-cut thresholds, leading to variability in practice.

One of the most cited barriers is the fear of precipitating hypotension or renal injury during fluid removal. Many critically ill patients remain on the edge of hemodynamic instability, and even small decrements in intravascular volume may tip the balance toward tissue hypoperfusion. While studies suggest that negative fluid balance is associated with improved survival, individual tolerance to deresuscitation is highly variable, and the risk of under-

perfusion is a genuine concern. This is especially pertinent in patients with septic shock, where ongoing vasoplegia may mask volume depletion, or in those with significant right ventricular dysfunction, where aggressive fluid removal can precipitate circulatory collapse.

Another layer of complexity is introduced by heterogeneous ICU populations. Patients with pre-existing comorbidities—such as heart failure, cirrhosis, or chronic kidney disease—often exhibit markedly different hemodynamic responses to fluid withdrawal. For example, heart failure patients may benefit from earlier and more aggressive deresuscitation due to their vulnerability to pulmonary edema, while patients with CKD or on dialysis may require ultrafiltration rather than diuretics to safely achieve negative balance. This heterogeneity complicates the development of universal protocols and underscores the need for individualized, physiology-guided strategies.

A further, often overlooked challenge is the iatrogenic contribution of maintenance and non-resuscitative fluids. Medications, nutrition, and electrolyte solutions can collectively account for several liters of fluid over the course of a week. Even as clinicians aim to restrict resuscitative fluids, these “hidden fluids” may contribute to cumulative overload. This phenomenon makes fluid stewardship more complex, requiring a multidisciplinary effort that includes pharmacy, nutrition, and nursing staff to minimize unnecessary volume.

The role of advanced monitoring also remains controversial. Tools such as transpulmonary thermodilution (to measure extravascular lung water), bioimpedance spectroscopy, or bedside ultrasound-based techniques can provide valuable guidance for fluid removal decisions. However, their availability is often limited to high-resource ICUs, and interpretation requires expertise. Many centers still rely on traditional parameters like central venous pressure or urine output, which are notoriously poor indicators of fluid status. The lack of standardized, widely applicable monitoring modalities hinders broader adoption of deresuscitation strategies.

Compounding these clinical challenges is the lack of standardized definitions for both fluid overload and de-escalation. Studies variably define fluid overload in terms of cumulative balance (e.g., >10% of body weight), central venous pressure, or extravascular lung water. Similarly, deresuscitation has been inconsistently defined, ranging from diuretic administration to achievement of negative fluid balance or net fluid removal with RRT. This heterogeneity in definitions hampers the comparability of research, limits meta-analytic synthesis, and ultimately creates uncertainty in clinical guidelines.^[31,14]

There is also ongoing debate about the optimal modality of deresuscitation. Diuretics remain the mainstay, but questions remain about the best drug combinations, dosing strategies, and monitoring parameters. Ultrafiltration offers an alternative, especially in patients with diuretic resistance, but its role outside of established indications for renal replacement therapy remains contentious. Concerns include not only technical feasibility and cost, but also the potential for excessive or too rapid volume removal, which can compromise hemodynamic stability.

Finally, the timing of de-escalation remains a central controversy. While evidence suggests that earlier negative balance (within 72 hours) is associated with better outcomes, clinicians are often reluctant to initiate deresuscitation until they are confident the acute shock state has resolved. This hesitation risks prolonging the window of harmful fluid accumulation. The lack of reliable markers to identify the precise transition point from resuscitation to stabilization further complicates decision-making, and ongoing research is attempting to fill this gap.

In sum, practical implementation of fluid de-escalation strategies requires navigating uncertainties around patient selection, timing, monitoring, and choice of intervention. Until

standardized definitions and robust clinical protocols are developed, practice will remain variable, reflecting the tension between evidence-based benefits and clinician apprehension in the face of critically ill, unstable patients.

Conclusion

Fluid de-escalation is a vital, yet underused, phase of fluid management in critically ill patients. While initial fluids remain lifesaving, unchecked fluid accumulation leads to harm. A thoughtful, monitored approach—employing conservative input, diuretics, controlled ultrafiltration, and adjunctive strategies—can restore fluid balance, hasten recovery, and potentially improve outcomes. As the evidence grows and consensus consolidates, de-escalation will take its place as a cornerstone of fluid stewardship in modern critical care.

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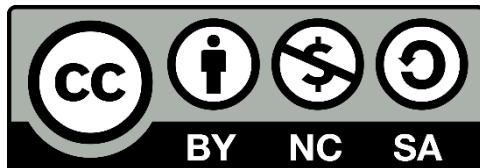
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